CLAIM OR CLAIMS

What is claimed is:

1. A peanut allergen having the tertiary structure shown and described herein.

2. The peanut allergen as recited in claim 1, wherein said peanut allergen is peanut allergen Ara h 1.

3. The peanut allergen as described in claim 2, wherein said tertiary structure consists of two sets of opposing sets of anti-parallel β-sheets in swiss roll topology with the terminal regions of the molecule consisting of α-helical bundles containing three helices apiece.

4. The peanut allergen as recited in claim 3, wherein there are numerous protease digestion sites throughout the length of this protein and the structure is so compact that potential cleavage sites are inaccessible until the protein is denatured.

5. The peanut allergen as recited in claim 4, wherein the formation of a trimeric complex and further higher order aggregation affords the molecule protection from protease digestion and denaturation and allows passage of the protein across the small intestine.

6. The peanut allergen as recited in claim 3, wherein the Ara h 1 IgE-binding epitopes are clustered on the surface of the molecule.

7. A tertiary structure for a peanut allergen Ara h 1 having the structure as shown and described herein.

8. The tertiary structure as recited in claim 7, wherein said structure is shown in at least one of Figures 3, 5, 11, 12, 31, 32, 38, 47, 48, and 49.

9. A method of producing the tertiary structure as recited in claim 7.

10. A method of using the nucleic acid sequences, proteins and peptides as shown and described herein.

11. Therapeutic compositions comprising proteins, nucleic acid molecules, antibodies, and

the like as shown and described herein.

12. The use of the therapeutic compositions of claim 11, to protect a host animal from allergic reaction.

13. A modified allergen epitope having at least one of the hydrophobic amino acids located in the middle of the epitope modified to prevent IgE binding.

14. The IgE binding peptides described in Table 1.

15. The alignment of the primary amino acid sequences of Ara h 1 and phaseolin A chain as shown in Table 3.

16. A DNA clone having homology with OP18, prothymosin alpha, and MM-1.

17. T-cell epitopes of Ara h 2 as shown and described herein.

18. The T-cell epitopes as recited in claim 17, wherein said epitopes are identified as epitope 1 (AA18-28), epitope 2 (AA45-55), epitope 3 (AA95-108), and epitope 4 (AA134-144).

19. The epitopes as recited in claim 18, wherein epitopes 1, 2 and 4 have overlapping sequences with Ara h 2 B-cell epitopes, whereas epitope 3 does not overlap IgE-binding epitopes.

20. A non-anaphylactic, T-cell directed immunotherapeutic peptide based on the epitopes of claim 18.

21. A peanut allergen Ara h 3 identified by using soy-absorbed serum IgE antibodies from peanut sensitive individuals and having significant sequence homology with the glycine family of seed storage proteins.

22. IgE-binding regions unique to soybean positive IgE binding, unique to peanut positive IgE binding, and common to both peanut and soybean IgE-binding regions as shown and

described herein.

23. An isolated recombinant peanut allergen designated Ara h 3.

24. An isolated nucleotide molecule encoding the peanut allergen designated Ara h 3.

25. A mutated peanut allergen protein having one or more IgE binding epitopes modified to reduce binding to IgE.

26. A method of treating an individual to reduce the clinical response to an allergen comprising administering to the individual a modified allergen which is less reactive with IgE in an amount and for a time sufficient to reduce the allergic reaction to the unmodified allergen.